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ABSTRACT

To investigate the relation between low level lead absorption and mild mental retardation, hair lead concentrations were compared in a group of 40 mildly retarded children "etiology unknown" with a control group of 20 children. Children with probable cause for retardation were excluded from the sample as were children with a history of lead poisoning. The group of mentally retarded children "etiology unknown" had statistically significant raised hair lead concentrations. It was concluded that a reexamination of lead poisoning concentrations is needed, because concentrations of lead previously thought harmless may now have to be considered noxious and viewed as an etiological factor in mild mental retardation. (Author/SB)

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LOW ELEVATED LEAD LEVELS AND MILD MENTAL RETARDATION

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Running Head: Low lead

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"Low Elevated Lead Levels and Mild Mental Retardation"

Many studies have confirmed that "lead poisoning" can cause severe mental retardation and neuropsychologic dysfunctions in children. It remains to be determined whether a smaller amount of lead, e.g., elevated but non-encephalopathic concentrations--can be an aetiological factor in mental retardation, especially in those mild and borderline cases where the aetiology is unknown.

Several studies have reported the association between lead concentrations much below those causing encephalapothy and central nervous system dsyfunctioning but evidence for a relationship between low concentrations of lead and intellectual dysfunctioning has not clearly emerged.

A major reason for discrepant conclusions in earlier studies is that the research designs with few exceptions, have used blood lead concentrations as an index of previous exposure. Recent studies have indicated however, that blood lead is not a reliable index of past absorption or toxicity per se (Chisolm, 1976; Vitale et al., 1975). Blood lead is an index of recent exposure, and it may return to normal levels even though exposure was excessive. Errors are likely to occur, therefore, if blood lead is relied on to classify subjects after exposure has abated (Needleman et al., 1979).

In this study lead concentrations were determined via hair samples and atomic absorption spectroscopy. Scalp hair

has several characteristics of an ideal tissue for epidemiologic study in that it is painlessly removed, normally discarded, easily collected, and its contents can be analyzed relatively easily (Hammer, Finklea, Hendricks, Shy & Horton, 1971). Trace elements, in particular, are accumulated in hair at concentrations that are generally at least ten times higher than those present in blood serum, provide a continuous record of nutritional status and exposure to heavy metal pollutants and may serve as a probe of physiologic functions (Maugh, 1978).

The best results have been obtained with heavy metal pollutants such as lead. Numerous investigations world wide have shown that concentrations of lead in the hair provide an accurate and relatively permanent record of exposure, and that there is a strong correlation between concentrations in hair and concentrations in internal organs (Kyle & Pease, 1966; Schroeder & Nason, 1969).

The purpose of the present research was to study the relationship between hair lead concentrations and retardation in a group of mild and borderline mentally retarded children without demonstrable cause for their mental deficit. The following hypothesis was proposed: the group of retarded children would have hair-lead concentrations significantly higher than those of a control sample.

Methods

Subjects

All subjects in this study were drawn from four schools in the Middle Tennessee Region. These included a preschool developmental center for handicapped and non handicapped children, two elementary schools, and a high school (grades 7-12). All of the mentally retarded subjects were receiving special educational services.

Their diagnosis of retardation was based on an overall evaluation from a series of consultations by school psychologists, classroom teachers, and appropriate other specialists where indicated. The diagnostic evaluation included the administration of a standardized intelligence test. All of the retarded children in the sample had an IQ of 55-84 inclusive (The American Association of Mental Deficiency classifies an IQ of 55-69 as "mild mental retardation" and 70-84 as the "borderline range.")

Presence or Absence of Probable Cause

All of the children's school records which met the IQ criteria were reviewed to determine whether there was a known or highly probable medical reason for their retardation. On the basis of this review, children with "probable cause," e.g., Down's Syndrome, hydrocephaly, brain injury, were removed from the study. In addition, any child with a history of diagnosed lead poisoning was excluded from the

sample study. Only children with unremarkable medical histories for mental retardation were assigned to the "aetiology unknown" group.

The control subjects were randomly drawn from the general school population at the four schools. No IQ determinations were available for the normal comparison group, but it appears safe to assume a relative absence of mental retardation in a randomly drawn general school population. Furthermore, a survey of the teachers' reports obtained for each child indicated average academic achievement and learning ability.

Table 1 shows the relevant demographic data for the two resulting groups of subjects. There were no significant differences between the groups in age, socio-economic status, or in the sex and ethnic group distributions.

Insert Table 1 about here

Hair Collection

After obtaining parental permission, children were asked to submit a small sample of hair (about 40 mg.) for trace mineral analysis. Hair samples were collected by the researchers using stainless steel scissors and then submitted to Doctor's Data, Inc., a federally licensed hair analysis laboratory in West Chicago, Illinois, where they were analyzed by atomic absorption spectroscopy.

Results

The two groups of children were compared for hair-lead concentrations. As shown in Table 2 the mean concentrations for the retarded children with unknown aetiology were

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Insert Table 2 about here

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considerably above those of the control children. The retarded group had a mean hair-lead of 15.9 ppm. while the control group had a mean hair-lead of 9.16. The data was analyzed with the *t*-test for two independent samples design, yielding a statistically significant *t* value, ($t = 3.39$, 65, $P < .001$) indicating that the variation between the two means was unlikely to have occurred by chance.

Fifteen ppm. is considered to be the normally tolerated limit of hair lead (Doctor's Data, 1980). The distribution of lead concentrations in the two groups is shown in Table 3.

The retarded group had the largest number of children in the higher ranges, 15 ppm. and above. Forty-five percent of the retarded children had lead concentrations above the normally tolerated limit compared to approximately 9% in the control group.

Insert Table 3 about here

Discussion

While not establishing an aetiological relationship, the data of this study shows a clear association between raised but non-encephalopathic hair lead concentrations and mild mental retardation. The study's hypothesis was strongly supported by the data.

The confidence with which mild mental retardation can be attributed to past lead exposure depends upon the adequate classification of earlier lead exposure. The classification of lead exposure to hair lead levels has been validated in a number of earlier studies. Hair lead levels are elevated in children with chronic lead poisoning (Kopito, Byers, & Shwachman, 1967) and in children with increased lead burdens (Pueschel, Kopito, & Schwachman, 1972). Hair lead levels also vary in relation to the concentration of lead in house-dust (Creason, Hinners, Bumgauner, & Pinkerton, 1975) and in relation to location proximity to a major lead processor (Roberts, et al., 1974).

In the present study, three parents of "elevated" retarded subjects initiated further diagnostic testing to confirm the initial screening results of their children. Venous blood and urine samples were collected and their lead content determined. The results of these tests confirmed the children's increased lead burdens.

A number of limitations of the study must be considered. First, with no more than 40 children in the mildly retarded

group and 27 children in the control group, this is a rather small study and doubts may arise on how representative the findings are, especially in respect to the controls.

Second, the determination of probable cause for retardation from reviewing school records is subject to errors common to retrospective studies in general. However, the most common errors in these studies are those of omission rather than commission. It is unlikely that a school record noting brain injury or a history of rubella is inaccurate; rather the error would occur because of absent data on medically significant factors, e.g., prenatal and perinatal factors. The relative inaccuracy implicit in the identification of the aetiology unknown group, however, probably leads to a lower mean lead concentration in this group, since the unidentified probable cause cases who might be erroneously included would be predicted to show lead concentrations comparable to a normal population. Such a prediction is supported by an earlier study in which a mentally retarded sample with "probable aetiology" showed no significant difference in lead concentrations from those of a normal control sample, while a group of mentally retarded children "aetiology unknown" had statistically significant raised lead concentrations (David, McGann, Hoffman, Sverd, & Clark, 1976).

The implications of this report are obvious and disturbing. The data shows that elevated lead concentrations much lower than those associated with overt lead poisoning

may also cause neurologic damage albeit a type that is more difficult to define.

The findings of this study suggest that a reexamination of lead poisoning concentrations is needed, because concentrations of lead previously thought harmless may now have to be considered lead poisoning and viewed as an aetiological factor in mild mental retardation. Other studies have also recommended a reexamination of lead concentrations, having linked low elevated lead levels to mental retardation, impaired IQ, deficits in visual, fine, and gross motor coordination and hyperactivity--findings previously reported as sequelae of overt lead poisoning (Lin-Fu, 1979).

At present there is no general agreement on the concentration of hair lead that should be considered dangerous. The findings of this study suggest that any elevation in hair lead levels above 15 ppm. must be regarded as potentially toxic.

Table 1 - Demographic Characteristics of 2 Groups of Children

	N	Sex M/F	Age (yr.)		Ethnic Group	
			Mean ± S.D.	Range	Caucasian	Black
Mentally retarded, aetiology unknown	40	29/11	11.00 2.353	4-16	38	2
Control group	27	16/11	11.07 3.198	4-14	26	2

Children in each group came equally from social classes 4 and 5 as defined by Hollingshed and Redlich (1958).

Table 2 - Comparison of Hair-Lead Concentrations Among 2 Groups

Group	N	Hair-lead (ppm.) (mean t S.D.	T value*
Mentally retarded, aetiology unknown	40	15.90 ± 8.40	3.39**
Control	27	9.16 ± 7.29	

* two tailed
** $p < 0.001$

Table 3 - Distribution of Hair-Lead Concentrations for 2 Groups of Children

Group	Hair-Lead Range (ppm.)		
	2-15	15.1-30	30.1 and above
Mentally retarded, aetiology unknown	22	15	3
Control group	22	5	0

15 ppm. is considered the acceptable upper limit of hair lead.

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